Chapter 34

Dermatology

Introduction

Most pediatric dermatological conditions are not acute and may be managed through telemedicine specialty consultation if dermatological expertise is needed. This chapter includes guidelines for telemedicine and focuses on situations requiring expeditious treatment in which potential delays in consultation may be deleterious to the patient.

Teledermatology Consultation

Requesting a teledermatology consultation is relatively easy for members of the US military. The following components should be submitted via e-mail to the US Army consultation service (derm.consult@us.army.mil; be sure to remove any personal patient information):

- Multiple focused photographs of all lesions on the patient
  - Photographs are best taken in well-lit settings (natural sunlight is optimal) without use of the camera's flash
  - Use the camera's "macro" setting, usually indicated by a picture of a flower, for close-up photographs of individual skin lesions
- Basic dermatological history of a lesion or condition, including:
  - Onset
  - Evolution
  - Duration
  - Location
  - Symptoms such as itch or tenderness
  - Alleviating and exacerbating factors
  - Current skincare regimen, including hygiene and topical preparations
  - Associated systemic symptoms
- Past medical history, including:
  - Medications and allergies

- Previous skin disease or cancer
- Systemic disease
- Evidence of atopic background (eg, eczema, asthma, allergies)

- Family/social history, including:
  - Skin diseases in other family members
  - Similar skin symptoms in close contacts
  - Pertinent environmental exposures, such as climate, pets, chemicals, harsh soaps, abrasive brushes, or radiation
- If basic laboratory facilities are accessible, a Gram stain, potassium hydroxide preparation, or Tzanck smear may immediately elucidate bacterial, fungal, or viral infections, respectively. If the facility is not equipped for these bedside tests, report laboratory test results that *have* been obtained, such as a complete blood count, and cultures or tissue specimens
- When describing the patient's physical examination, begin with vitals (especially fever) and general appearance
  - Remember that a thorough skin examination necessitates that the patient be fully disrobed and inspected from head to toe (eg, a common mistake to avoid is failing to examine the feet when a patient presents with hand dermatitis)
  - When describing the skin examination, use the following descriptive terms to aid communication with the dermatologist:
    - Primary skin lesions
      - Papule: an elevated, solid lesion up to 0.5 cm in diameter
      - Plaque: a circumscribed, elevated, superficial, solid lesion > 0.5 cm in diameter
      - Macule: a circumscribed, flat discoloration up to 0.5 cm in diameter
      - Patch: a circumscribed, flat discoloration > 0.5 cm in diameter
      - Nodule: a circumscribed, elevated, solid lesion > 0.5 cm in diameter; a larger, deeper papule
      - Pustule: a circumscribed collection of pus (cloudy, free fluid, and leukocytes)
      - Vesicle: a circumscribed collection of free, clear fluid

up to 0.5 cm in diameter
        - Bulla: a circumscribed collection of free fluid > 0.5 cm in diameter
        - Wheal (hive): a firm, edematous, transient plaque, which lasts hours at most
    - Secondary changes
        - Crust (scab): collection of dried serum and cellular debris
        - Scale: excess dead epidermal cells, thickened stratum corneum
        - Erosion: a partial focal loss of epidermis
        - Ulcer: a full-thickness, focal loss of epidermis and dermis (deeper than erosion, heals with scarring)
        - Excoriation: an erosion caused by scratching, often linear
        - Atrophy: depression in the skin resulting from thinning of the epidermis or dermis
        - Lichenification: thickened epidermis induced by scratching (skin lines are accentuated)
    - Special skin lesions and descriptors
        - Telangiectasia: dilated superficial blood vessels (blanchable—empty completely with compression)
        - Petechiae: circumscribed deposit of blood < 0.5 cm in diameter (not blanchable)
        - Purpura: circumscribed deposit of blood > 0.5 cm in diameter (not blanchable)
        - Erythema: an area of uniform redness that blanches with pressure
    - Other important descriptors include:
        - Color (eg, erythematous, violaceous, hemorrhagic, dusky, skin-colored, blanchable/nonblanchable, beefy red, yellow, etc)
        - Size (eg, 4 mm, 2 cm, etc)
        - Shape (eg, ill-defined oval, well-defined linear streak)
        - Specific location (eg, generalized over arms and legs but spares the trunk, face, and palms or soles)
        - A complete description also includes some comment on the mucous membranes (mouth, eyes, genitals, etc) and skin appendages (hair, nails)
- A response should be sent from a dermatologist within 24

hours (often in as few as 6–8 h)

Febrile Child with Rash

Many inflammatory diseases and viral exanthems may give this type of clinical picture. The following diagnoses include the majority of diseases that need to be recognized and treated emergently:

- Rickettsial diseases
  - Rocky Mountain spotted fever, Mediterranean spotted fever, typhus, and others
  - Typically present with skin eruption, fever, headache, malaise, and prostration
  - Transmitted by ticks, fleas, or body lice
    - Original tick bite may be a clinical clue, and often morphs from inflamed papule to necrotic eschar (tache noir)
    - The skin eruption tends to become petechial as it progresses
  - Diagnosis is based on which diseases may be endemic, the clinical presentation, and indirect fluorescent antibody testing, which may be confirmed by Western blot
  - It is imperative, especially for the spotted fever group of rickettsial diseases, to treat preemptively with doxycycline (2.2 mg/kg/dose bid intravenous [IV]; maximum dose 200 mg/day) to prevent mortality (Figure 34-1)
- Meningococcemia
  - Typically presents acutely, with fever, chills, hypotension, and meningitic symptoms (eg, neck stiffness, photophobia)
  - Half to two thirds of patients develop a petechial or ecchymotic eruption, primarily on the trunk and lower extremities, as well as petechiae on the eyelids and acral surfaces (Figure 34-2)
  - The petechial eruption tends to progress to hemorrhagic bullae or frank necrosis
  - Diagnosis is made on clinical suspicion because treatment needs to be rapid
    - Blood cultures growing the gram-negative diplococci of *Neisseria meningitidis* confirm the diagnosis
    - Treat these patients emergently with aqueous penicillin G (100,000–400,000 units/kg/day divided every 4–6 h,

maximum dose of 24 million units/day) or ceftriaxone (100 mg/kg/day divided every 12 h IV, maximum dose 2g/day) for 7 days
    - Close contacts should receive prophylactic rifampin (children) or ciprofloxacin (adults)
- Measles (rubeola)
  - Extremely infectious and spread by respiratory droplets
  - A prodrome of high fever, malaise, conjunctivitis, cough, and coryza (head cold symptoms) is followed by a maculopapular eruption (ie, generalized erythematous macules and papules), which begins on the head and progresses down the body over 2–3 days (Figure 34-3)
  - Koplik spots (white papules on an erythematous background on the buccal mucosa) are pathognomonic and appear during the prodrome, lasting 3 days (Figure 34-4)
  - Complications of measles include pneumonia, encephalitis, lymphopenia, thrombocytopenic purpura (rarely also disseminated intravascular coagulation [DIC]), and fetal death in pregnant women
  - Diagnosis is clinical, and treatment is supportive (rest, antipyretics, analgesics)
  - Affected children should be given vitamin A (2 doses of 200,000 units 24 h apart) to reduce morbidity and mortality
    - Giving vitamin A supplements to the entire population at the start of an outbreak will significantly reduce mortality
- Scarlet fever
  - Caused by toxins of group A streptococcal infection
  - Primarily affects children < 10 years old and was often fatal before the era of antibiotics
  - Presents with sore throat (streptococcus pharyngitis or tonsillitis), malaise, headache, nausea, abdominal pain, and high fever; an erythematous, blanchable, sandpaper-like rash follows within 12–48 hours
    - The eruption looks like "a sunburn with goose pimples," and begins on the neck, groin, and axillae before spreading to the rest of the body (Figure 34-5)
    - Pastia sign, petechial linear streaks within flexural

creases, may also be seen
    - Palms, soles, and conjunctivae are typically unaffected
    - In addition to the pharyngitis, the child may have circumoral pallor, cervical lymphadenopathy, palatal petechiae, and white strawberry tongue, which later morphs into red strawberry tongue
  - Diagnosis is clinical, though a throat culture may be useful
  - Treatment with penicillin or amoxicillin should be initiated to prevent the development of rheumatic fever
  - Desquamation typically occurs 7–10 days after the eruption, and should not be cause for concern
  - Complications include otitis, peritonsillar abscess, pneumonia, myocarditis, meningitis, and arthritis, as well as the immune sequelae of glomerulonephritis and rheumatic fever
    - Consider measles, toxic shock syndrome, drug hypersensitivity, staphylococcal scalded skin, and Kawasaki disease in the differential
- Kawasaki disease (mucocutaneous lymph node syndrome; Figure 34-6) is typically seen in children > 5 years old and is diagnosed by the presence of fever for > 5 days plus four of the following criteria:
  - Polymorphous skin eruption
  - Stomatitis (injected pharynx, strawberry tongue, fissuring cheilitis)
  - Edema of the hands and feet
  - Conjunctival injection
  - Cervical lymphadenopathy
    - An erythematous, desquamating perianal rash is often an early sign
    - As the illness progresses over 10–20 days, fingers and toes may desquamate, starting around the nails
    - Diagnosis is clinical. Rapid treatment with IV immunoglobulin (IVIG), if available (2 g/kg given over 10–12 h, see package insert for specific administration guidance) and aspirin is imperative to prevent potentially fatal coronary artery aneurysm, which complicates up to 25% of untreated cases
- Toxic shock syndrome can be caused by either streptococcus

or staphylococcus
- Streptococcal toxic shock syndrome presents with fever, shock, multiorgan system failure, and usually soft-tissue infection, such as necrotizing fasciitis
  - Streptococcal toxins have direct effects on the major organs, leading to shock and ultimately a mortality rate of 30%
  - Do not wait for cultures; treat emergently with fluids and pressors (for hypotension), clindamycin (40 mg/kg/day divided every 6–8 h), and surgical debridement of any soft-tissue infection source
- Staphylococcal toxic shock syndrome, caused by an exotoxin of *Staphylococcus aureus*, is typically associated with staphylococcal wound or catheter infections, or surgical or nasal packing
  - Patients present with sudden onset of high fever, myalgias, vomiting, diarrhea, headache, and pharyngitis; progression to shock can be rapid and fatal
  - Patients typically develop a diffuse scarlatiniform exanthem, erythema, and edema of the palms and soles, strawberry tongue, hyperemia of the conjunctiva, and a nonpitting generalized edema
  - Treat with supportive care and penicillinase-resistant antibiotics; remove any nidus for infection, such as nasal packing, tampons, and catheters
  - When considering toxic shock syndrome, also include Kawasaki disease, scarlet fever, staphylococcal scalded skin, toxic epidermal necrolysis, systemic lupus, rickettsial spotted fevers, and leptospirosis in the differential diagnosis
  - Desquamation of the palms and soles typically follows toxic shock syndrome by 1–3 weeks

- Leptospirosis (Weil's disease, pretibial fever, Fort Bragg fever) presents with abrupt onset of chills, high fever, jaundice, renal disease (proteinuria, hematuria, azotemia), petechiae, and purpura on skin and mucous membranes
  - Death occurs in 5%–10% of patients
  - Anicteric leptospirosis may also occur, and is sometimes termed "pretibial fever" because of characteristic shin pain

associated with erythematous rash most marked on the shins
    - Conjunctival suffusion, hemorrhage, ocular pain, photophobia, and intense headache may be other clues to diagnosis
    - Acquired from contact with water or soil contaminated with urine or tissues of infected animals (often rats, dogs, and cats)
    - Diagnosis may be made by finding the spirochetes of Leptospira in the blood (using smear, culture)
    - Treatment with penicillin (IV if disease is severe) and/or doxycycline during the first week of illness will shorten the disease duration

Bullae, Erosions, and Desquamation in Neonates and Young Children

The differential diagnosis for bullae in the neonate is vast and may include a variety of incurable genetic disorders that must be managed with intensive supportive care. For this reason, in the deployed setting, it is best to determine if the bullae are related to something acute or treatable, such as bullous staphylococcal impetigo or acute burn injury

- Bullous staphylococcal impetigo
    - Can be life-threatening in neonates and often occurs between the fourth and tenth days of life
    - Bullae may appear on any part of the body, often starting on the face or hands
    - Weakness, fever, and diarrhea may follow; sepsis may occur without antistaphylococcal antibiotics
- Staphylococcal scalded skin syndrome (Figure 34-7)
    - Preferentially affects neonates and young children
    - Begins with abrupt fever, skin tenderness, and erythema involving the neck, groin, and axillae
    - Over the next few hours to days, erythema progresses to cover more surface area, before ultimately evolving into a very superficial, generalized desquamation (more superficial and less severe than in toxic epidermal necrolysis)
        - Large sheets of epidermis separate
        - Nikolsky's sign is positive (the upper layer of the skin

detaches with application of lateral fingertip pressure to intact skin adjacent to a lesion)
    - The affected skin extends far beyond areas of actual infection (usually the pharynx, nose, ear, conjunctiva, skin, or septicemia)
  - Cultures are best taken from the mucous membranes
  - Rapid diagnosis can be made by examining frozen sections of a skin biopsy from a blister
    - In Figure 34-8, the superficial blister (just beneath the stratum corneum) is caused by a staphylococcal toxin to desmoglein 1, a protein that helps link keratinocytes together
    - This helps differentiate staphylococcal scalded skin syndrome from pemphigus vulgaris or toxic epidermal necrolysis, both of which may also present with superficial (Nikolsky-positive) blistering or denudation, but would show a slightly deeper split in the epidermis, histologically
  - Treatment includes IV fluids and cefazolin (50–100 mg/kg/day IV divided every 8 h, maximum dose 6 g/day; adjust for methicillin-resistant *S aureus* as necessary according to cultures)
- Neonatal herpes simplex virus (HSV) can also be life-threatening
  - Presents with vesicles in the newborn (Figure 34-9)
  - The majority of transmission occurs during delivery, and almost all patients present at 4–21 days of life
  - May be localized to skin and/or eye, involve the central nervous system, or be disseminated (encephalitis, hepatitis, pneumonia, coagulopathy)
    - Encephalitis or disseminated HSV can be fatal in up to half of affected neonates
    - More than half of survivors suffer neurological disability
  - Classic HSV presentation involves painful clusters of vesicles on an erythematous base; however, in over a third of cases, vesicles may not be seen
    - Clusters of erosions tend to remain where vesicles were located

- When these clusters coalesce, they leave behind an erosion or shallow ulcer with scalloped borders
  - Confirm this diagnosis with direct fluorescent antibody staining or a Tzanck preparation of smears obtained from the base of an unroofed vesicle, and with viral culture
  - However, if neonatal HSV is suspected, begin IV acyclovir (60 mg/kg/day IV divided every 8 h × 21 days) immediately
    - Central nervous system involvement may be detected by polymerase chain reaction of cerebrospinal fluid
- Bullous drug reactions can vary dramatically in severity, and many dermatologists consider drug-induced erythema multiforme, drug-induced Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN) to represent variations of the same disease process but on a spectrum of severity (Figures 34-10 and 34-11)
  - The definitions of SJS and TEN are somewhat arbitrary and overlap exists; usually SJS affects < 10% body surface area and TEN affects > 30% body surface area
  - The more widespread and severe the epidermolysis, the worse the prognosis and the more likely the process is drug-induced
  - The separation of skin layers in TEN is deeper than that in staphylococcal scalded skin syndrome (see above), with resultant denudation comparable to widespread burns. Accordingly, patients with TEN require intensive supportive care in an intensive care unit or burn center
  - Patients with SJS have a better prognosis than those with TEN, but whether the mucocutaneous denudation is due to TEN or SJS, offending drugs should be discontinued immediately and the patient should be transferred to an intensive care unit for careful fluid management, wound care, nutritional support, and sepsis precautions
    - Early ophthalmologic intervention is needed to

avoid ocular scarring and blindness
- The use of systemic corticosteroids and IVIG for either disease is controversial

▹ Erythema multiforme (minor) typically occurs in the spring or fall, and is often associated with HSV or, less commonly, *Mycoplasma pneumoniae*
- The lesions can have many different morphologies (hence "multiforme"), but are often targetoid in nature and can be acral in location (Figure 34-12)
- Most cases are self-limited and do not require treatment
- Daily suppressive anti-HSV therapy may be considered if the erythema multiforme recurs frequently

◦ Severe contact dermatitis can also present with widespread erythema and bullae
- ▸ Even when severe and widespread, contact dermatitis typically displays a patterned distribution
- ▸ Very pruritic
- ▸ In severe cases, patients may require a 3–4 week taper of oral corticosteroids
- ▸ Encourage good skin hygiene (at least twice daily gentle cleansing followed by bland emollient, such as petrolatum)
  - ▹ Antistaphylococcal antibiotics may also be necessary in cases of secondary infection (suggested by tenderness, expanding honey-colored crusts, and a positive bacterial culture)

◦ Atopic dermatitis (eczema)
- ▸ A common inflammatory skin disease in childhood characterized by intense pruritus and cutaneous inflammation
- ▸ In addition to affecting quality of life, atopic dermatitis can lead to disruption of the skin barrier and increase susceptibility to infection
- ▸ Associated with asthma and seasonal allergies (the atopic triad)
- ▸ Exacerbating factors may include irritants (soap, overwashing, drying chemicals) and allergens (skin contact

is far more detrimental than contact with foods or aeroallergens)
- ▹ Presentation in infants and toddlers: erythematous papules and plaques often with overlying excoriation, crusting, and serous exudates; distribution is usually over the scalp, forehead, cheeks, trunk, and extensor surfaces of the extremities
- ▹ Presentation in children and adults: dry, intensely pruritic papules and patches on flexor surfaces of the arms, neck, and legs. The dorsum of the hands and feet are often involved
  - ▪ Secondary changes, such as lichenification and postinflammatory hypopigmentation and hyperpigmentation are often seen
  - ▪ The differential diagnosis includes seborrheic dermatitis, psoriasis, pityriasis rosea, candidiasis, and keratosis pilaris

- ▸ Treatment
  - ▹ Avoid known allergens and irritants
  - ▹ Soak and smear: hydrate the skin by soaking in lukewarm water for 20 minutes; follow immediately (while the skin is still damp) by sealing in the moisture with emollient (ointment, cream, or thick lotion)
  - ▹ Moisturize frequently with less greasy lotions between soak-and-smear treatments
  - ▹ Use only mild soaps when necessary
  - ▹ Apply topical corticosteroids to localized plaques; taper off the steroids as the plaques clear
  - ▹ Use oral corticosteroids only in very severe cases (there is the potential for a rebound effect upon discontinuation)
  - ▹ Use topical or oral antibiotics directed against *S aureus* for superinfection

- ◦ Smallpox (variola major) has theoretically been eradicated since 1977, but is mentioned here due to its potential as a bioterrorism threat (smallpox was fatal in up to 40% of untreated patients)
  - ▸ Characterized by fever, severe headache and backache, and enanthem followed by exanthem

- Lesions progress simultaneously from erythematous macules to papules to vesicles, and finally pustules, which crust and collapse
  - This progression takes approximately 2 weeks
  - The lesions first appear on the palms and soles, then spread to the face and extremities (Figure 34-13)
  - By contrast, varicella (chickenpox) presents with lesions in varying stages of development (macules, papules, vesicles, crusts) and tends to involve the trunk and face preferentially
    - Varicella lesions evolve from vesicle to crust within 24 hours
    - Varicella can be identified at the bedside with a Tzanck smear, but a smear for direct fluorescent antibody staining and a viral culture confirm the diagnosis
  - Smallpox can be definitively diagnosed by viral culture, polymerase chain reaction, or electron microscopy; however, based on clinical suspicion, patients thought to have smallpox should be isolated, receive supportive care, and should be attended only by properly vaccinated healthcare workers
  - Smallpox is spread by respiratory route and patients are infectious from the onset of enanthem until about 10 days after the eruption begins (lesions are crusted)
  - Patients with varicella should also receive supportive care, but 20 mg/kg tid acyclovir over 5 days may speed recovery if initiated within 24 hours of the appearance of the eruption
    - Acyclovir should always be given to children > 13 years old and to any child with a history of atopic dermatitis
    - **Aspirin should never be given to varicella patients because of the risk of Reye's syndrome (fulminant hepatitis and encephalopathy)**

- Purpura in children
  - Henoch-Schönlein purpura (anaphylactoid purpura or allergic vasculitis; Figure 34-14) is the most common leukocytoclastic vasculitis in children ages 4–8 years old

(predominantly males)
- The trigger is often viral infection or streptococcal pharyngitis, as well as food, drugs, and lymphoma
- The rash is characterized by palpable purpura on the arms, legs, and buttocks
  - Patients can also have mild fever, abdominal colic, bloody stools, and arthralgias
  - Renal involvement with microscopic or gross hematuria is seen in half of patients, and a small percentage of patients go on to develop renal failure
  - Fatal pulmonary hemorrhage complicates rare cases
- Diagnosis is clinical
- Typically lasts 6–16 weeks
- Treatment is supportive, though oral corticosteroids can be given for abdominal pain
  - IVIG can be used in extreme cases
  - Nonsteroidal antiinflammatory drugs should be avoided (they may exacerbate renal or gastrointestinal problems)

◦ Purpura fulminans is a rapid, dramatic, and often fatal reaction seen in children (Figure 34-15)
- The appearance of widespread ecchymoses on the extremities, followed by frank acral necrosis, is characteristic
- DIC typically follows an infectious process, such as scarlet fever, meningitis, pneumonia, or superinfected varicella
- Treatment is supportive and best managed in the intensive care unit
- Many patients die from this complication and others require amputations

• **Necrosis in children**

◦ Necrotizing fasciitis, or "flesh-eating bacteria," is most commonly caused by β-hemolytic streptococci, though other organisms, both aerobic and anaerobic, can be causative

◦ Bacteria typically enter through a surgical or puncture wound, but necrotizing fasciitis may also occur de novo

- Within 24–48 hours, as the bacteria spread along fascial planes and destroy tissue, the involved skin progresses from red, painful edema to dusky blue, anesthetic skin, with or without serosanguineous blisters
- An early clue is tenderness outside the area of cellulitis
- By the fourth or fifth day, the purple areas become gangrenous
- Patients present in severe pain and are often hypotensive with leukocytosis
- Mortality is around 20%
- Prompt surgical intervention is necessary to conserve tissue and preserve the life of the patient
- Neonatal necrotizing fasciitis most commonly affects the abdominal wall and incurs a higher mortality rate than fasciitis in adults

- **Disseminated intravascular coagulation**
  - Presents with widespread intravascular coagulation and, in two thirds of patients, skin eruption of petechiae and ecchymoses with or without hemorrhagic bullae
  - Purpura fulminans may supervene (see above)
  - Can be due to a number of disorders, including sepsis, arthropod venom, obstetric complications, and acidosis
  - Supportive care and replacement of appropriate coagulation factors (including vitamin K and IV heparin) are necessary for these patients
- **Anthrax**
  - May pose a bioterrorism threat, but otherwise typically occurs from the handling of animal hides or from contact with infected animals (alive or dead)
  - Patients present with a rapidly necrosing, painless eschar with suppurative regional adenitis (Figure 34-16)
  - Anthrax may be cutaneous (most common), inhalational, or gastrointestinal
  - Cutaneous anthrax begins as an inflamed papule at the inoculation site
    - The papule progresses to a bulla with surrounding edema
    - The bulla ruptures to leave behind a dark brown or black eschar, surrounded by vesicles over a red, hot, swollen base

- Although regional lymph nodes may be tender, the anthrax lesion itself is painless
- In about 20% of untreated cases, patients may develop high fever, prostration, and death over days to weeks
- Diagnosis is made by identifying the gram-positive bacillus in smears or culture (Figure 34-17)
- Treat early with ciprofloxacin or doxycycline for 60 days
  - If the child is < 2 years old, treat intravenously
  - Asymptomatic exposed individuals should be given 6 weeks of prophylactic doxycycline or ciprofloxacin

Further Reading

1. **www.learnderm.org**. Tutorial takes approximately 1½ hours and encompasses the essentials of the skin examination, including describing morphology and recognizing patterns of distribution and configuration.
2. **www.visualdx.com**. From any US military computer, click on the login tab (US military has purchased access). Comprehensive source of skin lesion photos. May enter diagnosis or key words to search to get a visual differential.

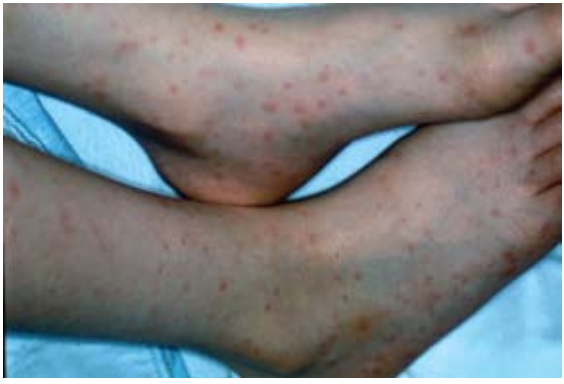
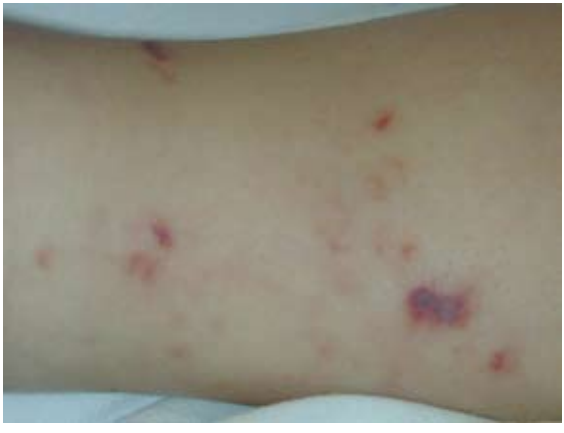


figure 34-1

figure 34-2



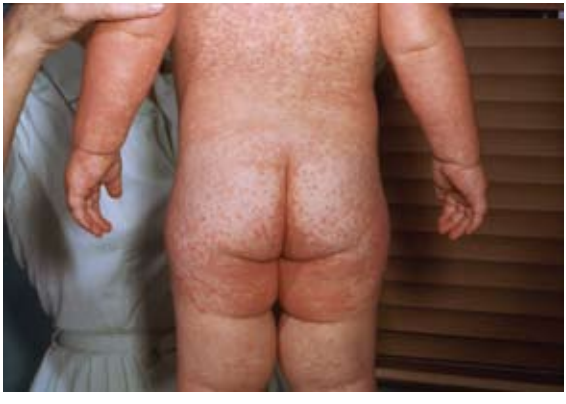


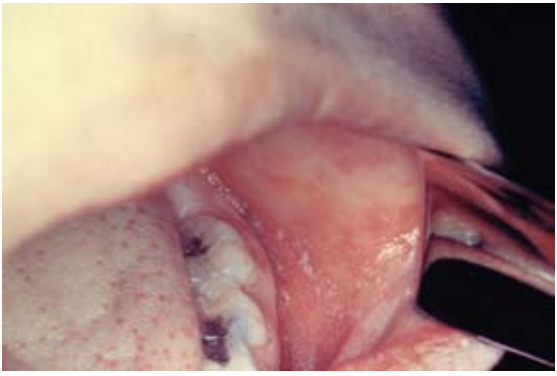
figure 34-3

Figure 34-1. This 9-year-old boy developed a red, partially blanching, papular eruption on his hands and feet, including his palms and soles, that progressed to the trunk over 3 days. He had a severe headache, high fever, arthralgias, and myalgias. His mother, who remembered removing a wood tick from her son's scalp 10 days earlier, also developed a rash, fever, and headache.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Bernard Cohen, MD, 2001. Image rmsf_1_010907.

Figure 34-2. This 7-year-old boy developed a severe headache, fever, and a palpable purpuric rash on his arms and legs. He complained of nausea and severe episodic pain in his arms and legs. A blood culture grew *Neisseria meningitidis* within 24 hours, and broad-spectrum antibiotic coverage was switched to high-dose penicillin. He was afebrile and feeling much improved 2 days later. Note the irregularly shaped and angulated character of the purpuric papules, some of which showed central necrosis.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Bernard Cohen, MD, 2002. Image meningococcemia_1_020403.

Figure 34-3. This child with measles is showing the characteristic red blotchy rash on his buttocks and back during the third day of the rash. Measles is an acute, highly communicable viral disease with prodromal fever, conjunctivitis, coryza, cough, and Koplik spots on the buccal mucosa. A red blotchy rash appears around day 3 of the illness, first on the face, then becoming generalized.
Reproduced from: Centers for Disease Control and Prevention Public Health Image Library Web site. http://phil.cdc.gov. Accessed November 12, 2009. Image 4497.

figure 34-4



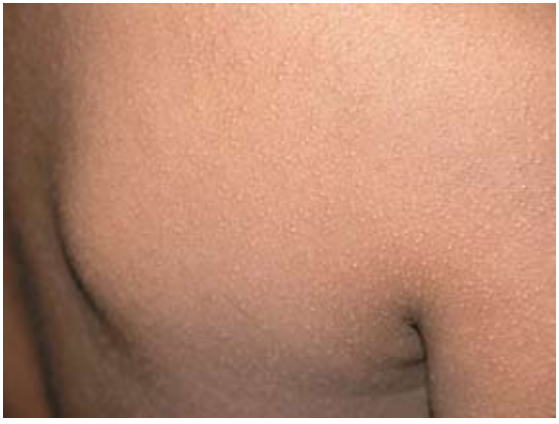


figure 34-5

figure 34-6

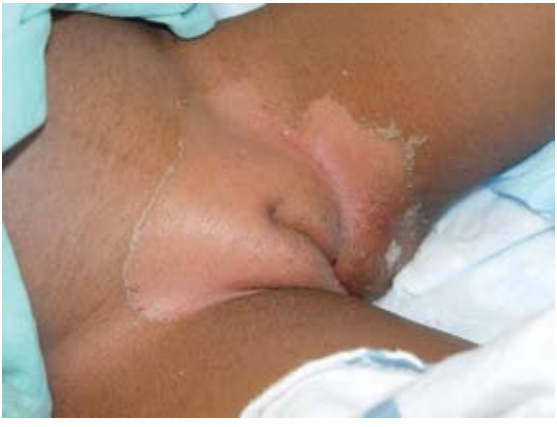


Figure 34-4. This individual with measles is displaying red Koplik spots on his buccal mucosa during the third day of the rash. Koplik spots occur 1–2 days before to 1–2 days after the cutaneous rash. Their presence is considered to be pathognomonic for measles, and they appear as punctate blue-white spots on the bright red background of the oral buccal (cheek) mucosa.
Reproduced from: Centers for Disease Control and Prevention Public Health Image Library Web site. http://phil.cdc.gov. Accessed November 12, 2009. Image 4500.

Figure 34-5. Scarlet fever. This 5-year-old boy had a 5-day history of sore throat and fever. He developed a generalized, fine, sandpaper-like rash on day 4 of illness. He also had red, swollen tonsils, and palatal petechiae. The case was prepared by University of Mississippi year 4 medical student, P Holland Alday.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Will Sorey, MD, 2008. Image scarlatina_1_081007.

Figure 34-6. This previously healthy 4-year-old girl developed high fever for 6 days, widespread scarlatiniform eruption, tender right anterior cervical adenopathy, conjunctival injection, red swollen hands and feet, strawberry tongue, and a peeling, scaly, red diaper dermatitis. She began receiving intravenous immunoglobulin 4 hours before the images were taken, and her mother thought that her symptoms were already improving.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Bernard Cohen, MD, 2004. Image kawasaki_2_040112.

figure 34-7

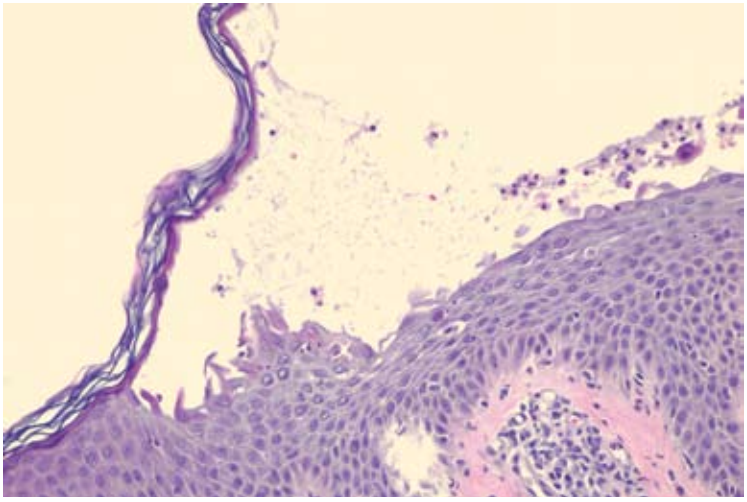


figure 34-8

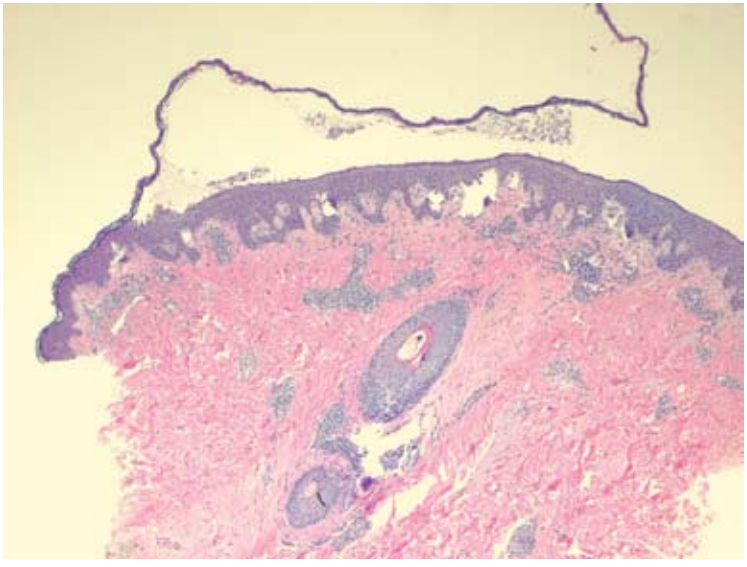
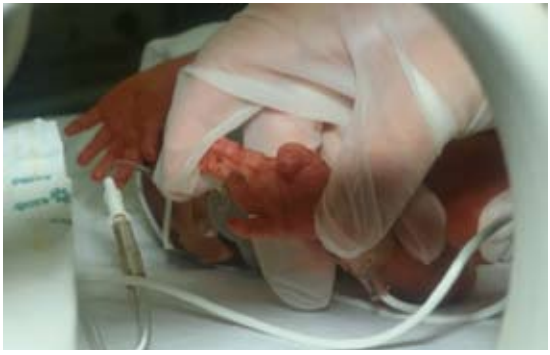


Figure 34-7. Staphylococcal scalded skin syndrome. This previously healthy 6-month-old girl developed a low-grade fever, increased fussiness, and generalized erythema with scaling, crusting, and fissuring accentuated around the mouth and skin creases. An erosion appeared after her mother placed her on the examining table, typical of a Nikolsky sign. Methicillin-sensitive *Staphylococcus aureus* grew from a skin culture taken from her nares. She did well on oral antibiotics, acetaminophen, and topical emollients, with drying of the crusts and clearing of the erythema in 2 days and diffuse desquamation 5 days later.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Bernard Cohen, MD, 2009. Image staphylococcal_scalded_skin_syndrome_4_090120.

Figure 34-8. Staphylococcal scalded skin syndrome. This 15-year-old patient developed an erythematous eruption over her face, neck, and trunk, which spread to her extremities. Denudation followed. This patient is a little old for staphylococcal scalded skin syndrome, but it may occur in adults. The histology includes a superficial blister in a subcorneal location, caused by a toxin to desmoglein 1. Because of the pathophysiology, the histologic differential diagnosis includes pemphigus foliaceus and bullous impetigo. Staphylococcal scalded skin syndrome is usually paucicellular, more so than in this case, but inflammation may ramp up in older lesions. Few acantholytic cells are evident in the cleft, as are a few inflammatory cells and serum.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Greg Hosler, MD, PhD, 2008. Image staph_scalded_skin_2_080801.

figure 34-9



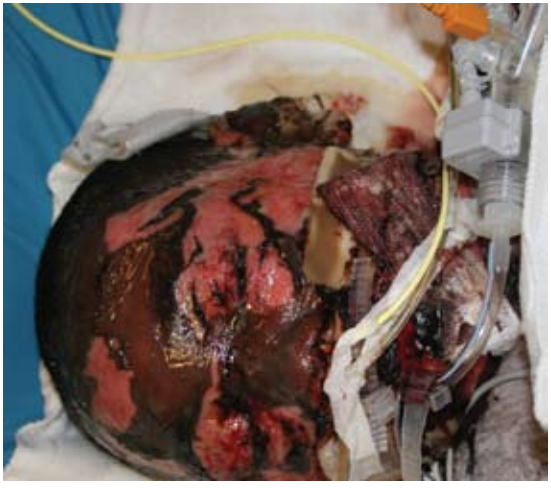


figure 34-10a

figure 34-10b

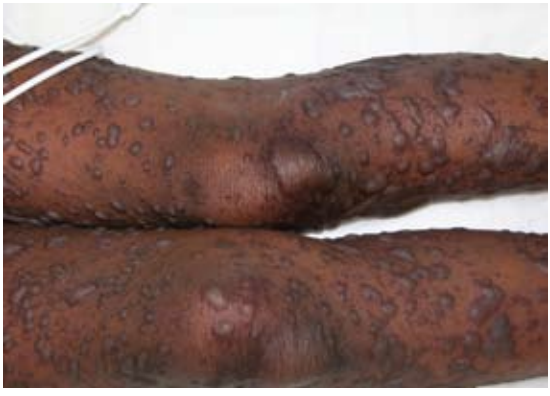


Figure 34-9. This premature infant had grouped vesicles on one hand and both feet at birth. A Tzanck smear was positive and a viral culture confirmed the diagnosis of neonatal herpes simplex virus infection.
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Figure 34-10. (**a**) This previously healthy 10-year-old boy had a seizure and was started on oral phenytoin. Three weeks later, he developed conjunctivitis and oral mucositis. Note the Nikolsky sign with widespread erosions triggered by general skin care.
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Figure 34-10. (**b**) Within 48 hours, he blossomed with confluent erosions above the waist and necrotic vesicles and bullae on the legs and feet.
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figure 34-11

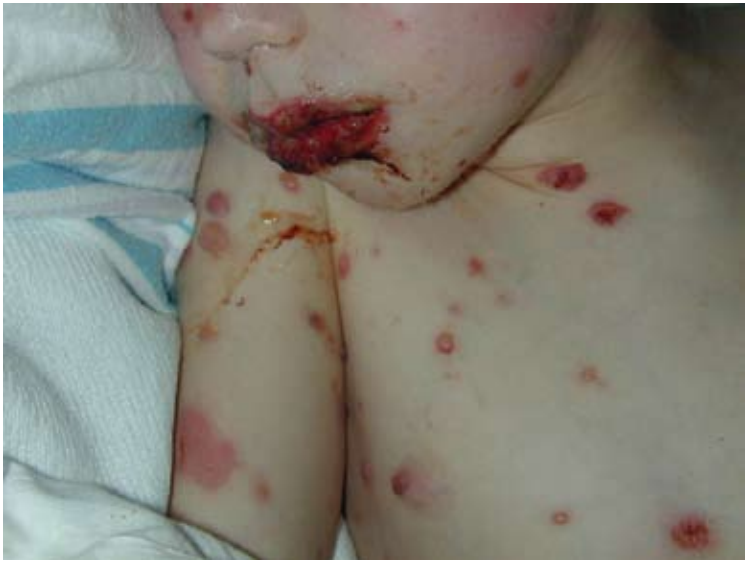

figure 34-12

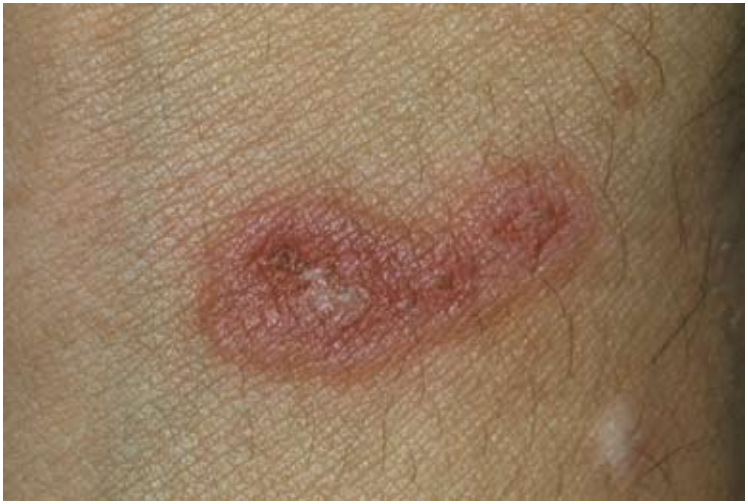

Figure 34-11. This 6-year-old boy developed a fever and pneumonitis with a left lingular infiltrate on chest x-ray. Ten days later, he complained of red itchy eyes, blisters in his mouth and red macules, some with central bullae, on the upper trunk, face, scalp, and extremities, including the palms and soles. Lesions progressed for 5 days before drying and healing with postinflammatory hyperpigmentation. He had no history of medication exposure before the illness, and mycoplasma titers were consistent with an acute infection.
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Figure 34-12. Erythema multiforme. A 16-year-old boy blossomed episodically with expanding red papules with central crust and bullous borders on his extremities, including palms and soles. Lesions appeared about twice a year and often involved the mouth as well. Skin biopsy of one of the arm lesions showed erythema multiforme, and culture of oral vesicles before one of the episodes yielded herpes simplex virus.
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figure 34-13

figure 34-14

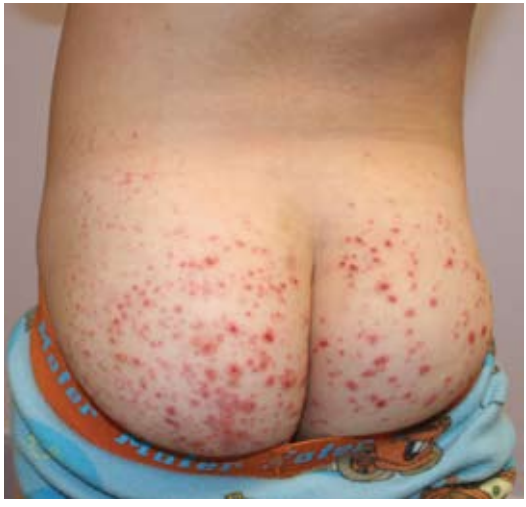
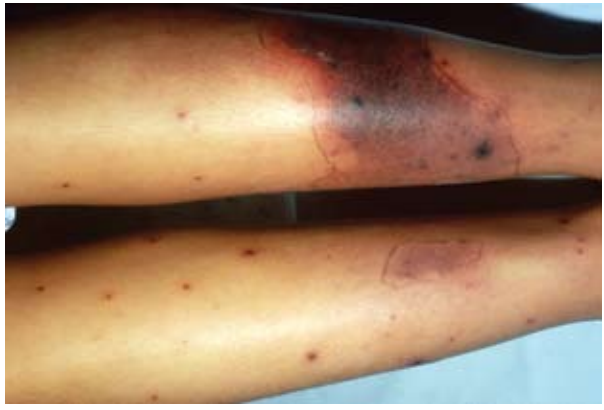


Figure 34-13. Smallpox. Depicted in this 1974 photograph is a Bengali boy who was an inhabitant of a Bangladesh village. As evidence from this image, he had sustained the ravages of smallpox, with the classic maculopapular rash evident on his torso and arm.
Reproduced from: Centers for Disease Control and Prevention Public Health Image Library Web site. Photograph courtesy of Jean Roy. http://phil.cdc.gov. Accessed November 12, 2009. Image 10661.

Figure 34-14. This previously healthy 4-year-old boy developed palpable purpuric lesions on his extremities, face, and buttocks 2 weeks after an upper respiratory infection. He subsequently experienced swelling of his ankles and wrists, colicky abdominal pain, and hematuria. His skin eruption and associated symptoms recurred for 3 weeks before resolving without complications.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Bernard Cohen, MD, 2008. Image henoch_schonlein_purpura_10_080209.

figure 34-15



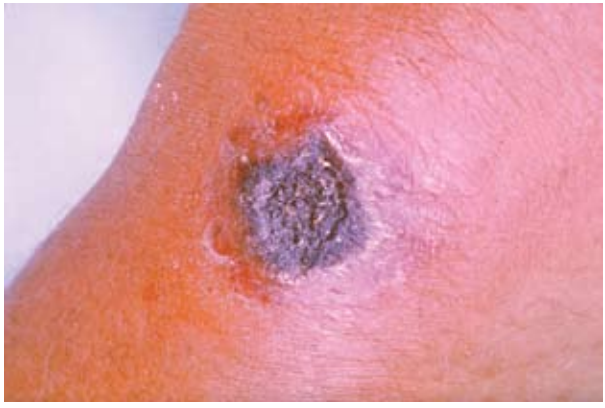


figure 34-16

figure 34-17

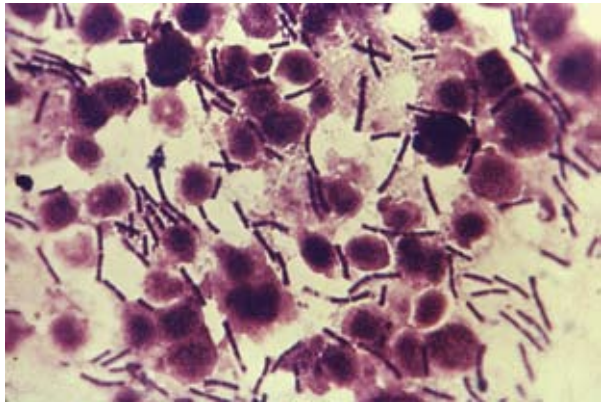


Figure 34-15. A healthy 10-year-old girl developed large expanding ecchymoses on her legs while she was recovering from varicella. She also bled into healing varicella crusts. Although she appeared well, she developed a high fever and laboratory signs of disseminated intravascular coagulation. A skin culture obtained from the crusted papules in the large ecchymoses grew Group A β-hemolytic streptococcus. She was treated with parenteral antibiotics and fluid resuscitation, and fortunately recovered without complications. The day after her admission to the hospital, a new systolic murmur was noted, and a subsequent echocardiogram showed a large clot in the ascending aorta. This also resolved uneventfully.
Reproduced with permission from: Dermatlas.org. http://www.dermatlas.org. Copyright Bernard Cohen, MD, 2001. Image purpurafulminans_1_010729.

Figure 34-16. Anthrax lesion on the skin of the forearm caused by the bacterium *Bacillus anthracis.* Here the disease has manifested itself as a cutaneous ulceration, which has begun to turn black, hence the origin of the name "anthrax," after the Greek name for coal.
Reproduced from: Centers for Disease Control and Prevention Public Health Image Library Web site. Photograph courtesy of James H Steele. http://phil.cdc.gov. Accessed November 12, 2009. Image 2033.

Figure 34-17. Photomicrograph of *Bacillus anthracis*; anthrax.
Reproduced from: Centers for Disease Control and Prevention Public Health Image Library Web site. http://phil.cdc.gov. Accessed November 12, 2009. Image 1811.